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Adynamic bone disease: an update and overview.

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Adynamic bone disease (ABD) is a variety of renal osteodystrophy characterized by reduced osteblasts and osteoclasts, no accumulation of osteoid and markedly low bone turnover. It has been found in a relatively high percentage of patients on dialysis, either peritoneal or hemodialysis, but also in CKD patients on conservative treatment. The histologic pattern of ABD is generally associated to low levels of PTH. However, PTH serum levels in CKD are generally higher than normal even when associated to ABD. Therefore, it is felt that, basically in uremia, bone tissue is resistant to PTH, so that a relative reduction of its levels is able to induce the emergence of a low turnover state. Several factors theoretically responsible for skeletal resistance to PTH, and able to slow bone turnover have been considered. Among these are downregulation of PTH receptors in bone cells, increased levels of osteoprotegerin, decreased production and circulating levels of bone morphogenetic proteins, the peripheral effect of leptin and also a possible effect of increased N-terminal truncated PTH molecular species, which have been found to counteract the whole molecule, PTH 1-84 on the bone. In conclusion, ABD should probably be considered a skeletal condition induced by overtreatment of secondary hyperparathyroidism and not a disease. However, its development reveals

a deranged ability of uremic bone to maintain a normal bone turnover, when PTH serum levels decrease beyond relatively low levels, which would be considered normal in the general population.

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